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EDITORIAL

Vasovagal syncope: poorly understood, but well handled

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This editorial refers to 'Cardiac autonomic disturbances in patients with vasovagal syndrome: comparison between iodine-123-metaiodobenzylguanidine myocardial scintigraphy and heart rate variability' by G. Kochiadakis *et al.*, on page 1352

In this issue of the Journal, Kochiadakis and co-workers from Heraklion University Hospital in Athens report on autonomic regulation in patients with vasovagal syncope (VVS) diagnosed with tilt table testing.[1] They measured heart rate variability (HRV) and assessed cardiac sympathetic innervation using 123-I-meta-iodobenzylguanidine (MIBG) scintigraphy. The main finding of this study was that adrenergic cardiac innervation showed defects in the left ventricle in patients with VVS. This finding was not associated with the type of response (cardioinhibitory or vasodepressor) during tilt table testing. However, the study critics may object that the infrequent blood pressure measurements rather than continuous non-invasive blood pressure measurement during tilt table testing did not allow the accurate classification of the response. In contrast to this finding, HRV, as a marker of the resulting efferent vasomotor activity at the level of the sinus node, did not show any significant difference between patients and control subjects when measured the day after the tilt test.

Thus, on the one hand, the authors report an interesting new finding pointing to a potential pathoanatomical substrate involved in the complex mechanism of VVS, whereas on the other hand a well-known method, claimed to monitor the sympathovagal activity, fails to show any difference.

There has been a great enthusiasm about HRV as an easily obtainable and simple non-invasive measure of the influence of the autonomic nervous system after the early paper by Akselrod *et al.*[2] The method was applied to short-term recordings in the laboratory as well as ambulatory 24 h electrocardiographic (ECG) recordings, and algorithms for computing HRV in both the time domain and power spectral analysis have soon become commercially available. This new tool was used to investigate the efferent autonomic control of the sinus node in various conditions and to predict future arrhythmic events in diseased hearts. Despite the availability of ECGs and many attempts in prospective studies, there has not been any clinical breakthrough for HRV-derived indices to have an independent prognostic value.[3] Regarding the applicability of HRV as a physiological measure of cardiac autonomic control, it is characteristic that the authors cite earlier studies from 1994[4] and 1986.[5] These publications refer to a different method of quantifying the HRV than that utilized in the present study, namely power spectral analysis. It would have been interesting to correlate the sequence of the cardiovascular response and the changes in HRV evaluated with power spectral analysis during the tilt table test, instead of time-domain analysis the day after the tilt test. In general, there appears nothing new in the literature, including the current paper, on HRV and the mechanisms of VVS.

It is, therefore, encouraging that Kochiadakis *et al.* attempted to find new clues to the VVS puzzle. With the use of MIBG, they have demonstrated that patients with VVS have an altered pattern of adrenergic cardiac innervation, suggesting persistent dysfunction of myocardial adrenergic innervation. Where HRV has shown only temporary changes during tilt in VVS patients,[6] but not in control state,[1] MIBG shows significant changes and this is present even in the normal state. This finding implies that VVS is not only a functional disorder but may have a pathoanatomical substrate. However, other aspects need to be addressed as bradycardia is not the only feature of VVS; control of blood pressure regulation, humoral factors, neural triggers, etc. are all of importance when trying to entangle the wide range of cardiovascular responses in VVS. Furthermore, it is important to appreciate that it may be a crude generalization to reduce all neurally mediated reflex syndromes to sharing the common pathway of events leading to bradycardia, hypotension, and loss of consciousness. Although the present finding needs to be reproduced and put into context of the complex mechanism involved in VVS, it represents a step forward in our understanding of VVS.

The European Society of Cardiology (ESC) guidelines from 2009[7] on diagnosis and management of patients with syncope is

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an excellent clinical tool, and it already has been followed by several clinical studies[8,9] on diagnosing and stratifying patients with syncope. Implementation of these guidelines and the establishment of dedicated syncope facilities are essential for the management of patients with this syndrome, whose underlying causes range from the benign isolated VVS to malignant arrhythmias.

In spite of advances in diagnosis and risk stratification of patients with syncope, treatment of VVS is often difficult and challenging, but a better understanding of the condition and precipitating factors may lead to better treatment. In this regard, the contribution by Kochiadakis and co-workers is welcome.

Conflict of interest: none declared.

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IMAGES IN ELECTROPHYSIOLOGY

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Superior vena cava obstruction due to pacemaker leads

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A 45-year-old woman was referred for a pulse generator change with insertion of a new ventricular lead. Given a wire would not advance into the right atrium, a venogram was performed. Injection of contrast demonstrated complete occlusion of the distal superior vena cava with extensive dilation of the hemiazygous system and collateralization into the inferior vena cava. The patient had no symptoms indicative of obstruction. In the coming years, this 'silent' complication will gain increasing attention as the number of patients requiring new leads will increase with ageing of the population, expansion of implant guidelines, and ongoing recalls.